# Food related cardiac diseases

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## Introduction

Cardiac disease is common in horses. Cardiac murmurs are frequently found and usually originate from normal cardiac function (physiological murmurs such as flow murmurs) or from valvular or congenital cardiac disease. Arrhythmias may be physiological, due to the horse's high vagal tone, or pathological. The latter may be caused by systemic disease or by primary myocardial injury.
Aetiological factors for myocardial injury are of traumatic (e.g. stretch due to cardiac dilatation), infectious, infiltrative, hyperthermic, ischemic, toxic, nutritional or idiopathic origin. Viral infection, such as Influenza, Equine Viral Arteritis virus, Equine Infectious Anaemia, African Horse Sickness, Equine Herpes Virus 1, Eastern Equine Encephalomyelitis, and bacterial infection, e.g. Streptococcus spp., Staphylococcus spp., Salmonella, Clostridium, Borrelia burgdorferi, piroplasmosis, Leptospirosis, Neorickettsia risticii, and also parasites as Strongylus vulgaris, Onchocerca, have been associated with myocarditis. Of all causes, food related disease is not very common but probably underdiagnosed. Food related disease may be a direct effect of the (lack of) normal nutrients or may be due to ingestion of or contamination by toxic products or plants. Nutritional imbalances leading to myocardial injury are usually related to vitamin E, selenium, copper and molybdenum. Food related intake of toxins that lead to myocardial injury are usually related to plants (Fox glove, Oleander, Adonis, Eonymus, Lily of valley, Yew, Rhododenderon,…), ionophore antibiotics (monensin, salinomycin, lasalocid), heavy metals, cantharidin toxicosis (blister beetle),…
The clinical signs, which are related to cardiac electrophysiological and contractile properties and to underlying disease, are very variable. Clinical signs range from subclinical to poor performance, exercise intolerance, syncope and sudden death. Clinical signs may be those of underlying systemic disease (fever, colic, weakness, respiratory signs, shock,…) or may be directly related to myocardial damage. Myocardial inflammation may result in arrhythmias, due to enhanced ectopy or impaired conduction, and in altered systolic or diastolic function. Enhanced ectopy results in atrial or ventricular premature beats, tachycardia or fibrillation whereby ventricular fibrillation is a fatal event. Enhanced or, more frequently, decreased ventricular contractile function may occur leading to exercise intolerance and weakness. In chronic cases myocardial fibrosis might develop.
In order to make a diagnosis, history is very important: duration of disease, possible contact with toxins, other animals involved,…. Horses generally avoid ingestion of toxic plants because of their bad taste. It should be clearly noted, however, that a single affected animal in a group does not preclude an intoxication. Indeed, some individuals have a less selective eating behaviour and are therefore more likely to ingest (bad tasting) toxic plants or products than others. Mowing plants without removing them from pasture increases the risk for intoxication because many plants become more palatable retaining their toxicity. Presence of toxic plants in hay or silage, often results in intoxication of multiple animals because the toxic plants are present throughout the hay and horses cannot avoid eating the toxic plants.
Physical exam may reveal dysrhythmias, a cardiac murmur, weak pulse, oedema. Electrocardiography (ambulatory, exercise, 24-hour) and cardiac ultrasound are crucial to evaluate cardiac function. While cardiac glycoside intoxication may result in increased inotropy (initially), most other toxins will produce contractile dysfunction. Complete blood count and biochemistry must be performed. Determination of cardiac troponin I or T is essential as it is the best parameter to detect myocardial injury. In case of suspicion of specific disease or intoxication, serum, urine samples, swabs,… should be taken for further diagnostic tests (serology, PCR, toxicology,…).
The horse should be rested for at least 6-8 weeks. Therapy aims to treat underlying disease and life-threatening dysrhythmias, improve cardiac function and treat heart failure. In case of intoxication, further access to the toxin must be avoided and activated charcoal or mineral oil should be administered by nasogastric tube in acute cases of oral intoxication. Horses must be closely monitored during the treatment. Horses that recover should always be re-examined before they are allowed to return to exercise to confirm that arrhythmias no longer occur. Indeed, presence of myocardial injury with ventricular arrhythmias is a known risk factor for sudden death.
Below some more information is given concerning specific conditions related to lack of nutrients or oral intake of toxic components that affect the myocardium.

## Vitamin E and/or selenium deficiency

Nutritional myodegeneration (white muscle disease) not only affects skeletal muscles but also myocardium. It is often associated with low vitamin E and/or low selenium levels. Muscle weakness, trembling, stiff and painful gait, recumbency and tachycardia and tachypnea are usually present. Diagnosis is based on clinical signs, increased muscle enzymes and cardiac troponin I or T levels, in conjunction with low vitamin E and/or selenium levels. Treatment includes pain management, fluids, NSAIDs and vitamin E and selenium administration. More information is given in a separate presentation about vitamin E.

## Atypical myopathy

Atypical myopathy (AM) is an acute, often fatal, rhabdomyolysis in grazing horses that mainly affects skeletal muscles and myocardium. The disease is caused by ingestion of the toxin hypoglycin A, that can be found in seeds, seedlings and leaves from the sycamore tree (*Acer pseudoplanus*) or the box elder tree (*Acer negundo*). Post mortem examinations have shown that myocardial damage also occurs. In affected animals cardiac troponin I or T levels are increased. Horses often show ventricular premature depolarisations or other pathological arrhythmias and a prolonged corrected QT (QTcf) interval. On ultrasound, myocardial wall motion is also abnormal. Treatment is mainly supportive and consists of pain management, fluid therapy, correction of acid-base and electrolyte status, administration of vitamin E and selenium. Also riboflavin and L-carnitine have been suggested to be beneficial. Survival rate is only about 25% and recovery may take weeks. In survivors, abnormal cardiac function may persist for several weeks. It is recommended to perform a cardiac re-evaluation prior to bringing the affected horse back into work.

## Cardiac glycoside intoxication

Intoxication with cardiac glycosides usually originates from ingestion of toxic plants or by overdosing with digoxin. Fox glove, oleander, Adonis aestivalis, convallaria majalis, apocynum and euonymus are known sources of cardiac glycosides. Ingestion generally occurs because of a lack of food, when plants are dried (e.g. cutting without removal from pasture) or when plants are present in hay or silage. Inhibition of Na-K-ATPase results in increased intracellular calcium. Initially this leads to conduction slowing and increased contractility. However, toxic doses lead to cell death and arrhythmias, which may be fatal due to ventricular tachycardia and fibrillation. Clinical signs include gastro-intestinal signs such as colic, sweating, diarrhea and dehydration. When cardiac glycoside intoxication is suspected, serum digoxin and digitoxin levels should be measured. Digoxin tests are readily available. The advantage of digitoxin is that is has a longer half live and might therefore be longer detectable in serum. Toxic plants contain a large number of different cardiac glycosides. Typically, the digoxin and digitoxin tests are rather specific (based on monoclonal antibodies) and there is only limited cross reaction with the broad range cardiac glycosides from plants. This means that the found digoxin or digitoxin levels might be fairly low, even below the therapeutic range, in horses that present severe, life-threatening intoxication. However, normal horse serum does not contain these molecules so positive levels support intoxication (unless the horse received digoxin treatment). The environment should be thoroughly checked for presence of toxic plants. Treatment consists of activated charcoal, perfusion, magnesium supplementation, and immediate therapy with lidocaine or phenytoin. After recovery, horses are rested for at least 8 weeks and exercise testing (ECG recording) is needed before resuming normal work.

## Ionophore intoxication

Ionophores, such as monensin, lasalocid, salinomycin and narasin, are used as anticoccidial drugs for poultry or growth promotors for ruminants. Horses are known to be very sensitive to ionophores, especially monensin and lasalocid, and poisoning results in severe signs. Accidental poisoning is usually the result of production or transportation errors or access to poultry or ruminant rations. Clinical signs include anorexia, depression, tachycardia and cardiac arrhythmias which are potentially lethal. These arrhythmias may vary from occasional ventricular premature depolarisations, to ventricular tachycardia, torsades des pointes and fatal ventricular fibrillation. On cardiac ultrasound a severely reduced contractile function is often present. The neuromuscular function may be affected resulting in abnormal gaits, ataxia, incoordination, weakness and paresis or paralysis. Due to severe myocardial damage troponin levels are markedly increased. The condition is often fatal especially in monensin intoxicated animals. Diagnosis is based upon history, clinical signs and identification of the drug in food or tissue. No specific antidote exists; treatment is supportive. Any further contact with potentially contaminated food should be avoided. Vitamin E should be administered and acts as protective factor against intoxication. Recovery is slow and survivors may present permanent lesions such as severe myocardial fibrosis. Again thorough cardiac re-evaluation is necessary before recovered horses can resume their normal level of activity, especially to confirm that ventricular arrhythmias are no longer present.

## Conclusion and take home message

Although not very common, food related cardiac disease should be included in the differential diagnosis of horses with anorexia, arrhythmias, tachycardia or gait abnormalities. As consequences can be severe and even lethal, early diagnosis and prompt treatment are essential in order to maximize survival and minimize risks for sudden death or permanent myocardial damage. Determination of cardiac troponin I or T levels is easy, quick and accurate to detect myocardial damage. Cardiac ultrasound allows to detect contractile dysfunction. Ambulatory ECG is needed to diagnose the origin of arrhythmias and determine the need for specific therapy.

## References


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